POST STROKE DEPRESSION:INCIDENCE AND ASSOCIATIONS

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CERTIFICATE

This is to certify that the dissertation titled "**POST STROKE DEPRESSION :INCIDENCE AND ASSOCIATIONS**" is the bonafide original work of **DR. M.KUMAR RAJA** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in SEPTEMBER 2006. The Period of study was from November 2004 to October 2005.

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DECLARATION

I, **DR. M.KUMAR RAJA,** solemnly declare that dissertation titled **"POST STROKE DEPRESSION :INCIDENCE AND ASSOCIATIONS"** is a bonafide work done by me at Govt. Stanley Medical College and Hospital

during 2004-2005 under guidance and supervision of my unit chief **Prof.S.NATARAJAN**., Professor and head of the department of medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

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CONTENTS

POST STROKE DEPRESSION
INCIDENCE AND ASSOCIATIONS

***INTRODUCTION*:**

A Stroke or cerebro vascular accident is defined as abrupt onset of a neurologic deficit that is attributable to a focal vascular pathology. Cerebrovascular diseases include

1.Ischemic stroke

2.Hemorrhagic stroke

3.Cerebrovascular anomalies------intra cranial aneurysm and arterio venous malformations. They are the major cause of disability. The incidence of stroke increases with age and the number of strokes is projected to increase as elderly population grows. The clinical manifestation of stroke are highly variable because of the complex anatomy of the brain and its vasculature.

Cerebral ischemia is caused by a reduction in blood flow that lasts longer than several seconds. Neurologic symptoms are manifest within seconds because neurons lack glycogen ,so energy failure is rapid. When blood flow is quickly restored ,brain tissue can recover fully and the patients symptom are only transient called transient ischemic attacks(TIA).Typically TIA signs and symptoms lasts for 5 to 15 minutes but by definition must lasts <24 hours.

If the cessation of blood flow lasts for more than a few minutes ,infarction results. Stroke has occurred if the neurologic signs and symptoms lasts longer than 24 hours. Infarction is usually caused by thrombosis of the cerebral vessels themselves or by emboli from a proximal arterial source or the heart. Cerebral hemorrhage produces neurologic symptoms by producing a mass effect on neural structures and vascular

insufficiency.

Rehabilitation after stroke begins as soon as the diagnosis of the stroke is established and as soon as any life threatening neurological or medical complications have been stabilised. DEPRESSIVE SYMPTOMS are common after stroke occurring in over 25% of patients. Stroke patients should be questioned and screened for depression. Depression is common following left hemisphere stroke especially in the frontal lobe, possibly caused by disruption of catecholamine pathways.

Treatment with antidepressants is often successful in ameliorating symptoms. Appropriate diagnosis and treatment of depression may bring substantial benefits to persons recovering from stroke by improving their medical status ,enhancing their quality of life and reducing their disability.

AIM OF THE STUDY

1. TO FIND OUT THE INCIDENCE OF DEPRESSION IN STROKE PATIENTS.

2. TO FIND OUT THE HEMISPHERE OF THE BRAIN COMMONLY ASSOCIATED WITH POST STROKE DEPRESSION

3. TO INCREASE THE AWARENESS AND IMPORTANCE OF ANTIDEPRESSANTS IN STROKE PATIENTS..

REVIEW OF LITERATURE

STROKE FACTS:

The most common site for a stroke to occur is in the distribution of middle cerebral artery which supplies much of the frontal ,temporal,and parietal lobes of the brain as well as the subcortical ganglia.

ETIOLOGY:

It is essential to establish the cause to reduce the risk of recurrence.

85% of CVA are infarct and remaining 15% of cases are hemorrhagic.

INFARCT:

1.Thrombosis:

Lacunar stroke[small vessel]

Large vessel thrombosis

Dehydration

2.Embolic occlusion:

Artery to artery:

[a]Carotid bifurcation

[b]Aortic arch

[c]Arterial dissection

Cardio embolic:

[a]Atrial fibrillation

[b]Mural thrombus.

[c]Myocardial infarction

[d]Dilated cardiomyopathy.

[e]Valvular lesions:----Mitral stenosis,

Mechanical valve,

Bacterial endocarditis .

[f]Paradoxical embolus:Atrial septal defect,

Patent foramen ovale.

Emboli from the heart most often lodge in the middle cerebral artery,the posterior cerebral artery or one of their branches. Infrequently anterior cerebral artery territory is involved.The most significant causes of cardioembolic stroke in most of the world are non rheumatic atrial fibrillation[Non valvular],Myocardial infarction, prosthetic valves,Rheumatic heart disease and ischemic cardiomyopathy.Non rheumatic atrial fibrillation is the most common cause of cerebral embolism overall. Patient with atrial fibrillation have an annual risk of stroke of 5%.

Other uncommon causes of stroke are:

1.HYPERCOAGULABLE DISORDERS:

Protein C deficiency.

Protein S deficiency

Anti thrombin III deficiency.

Anti phospholipid syndrome

Factor V Leiden mutation

Prothrombin G 20210 mutation.

Systemic malignancy.

Sickle cell anaemia.

Polycythemia vera.

Systemic lupus erythematosus.

Homocysteinemia.

Disseminated intra vascular coagulation.

Thrombotic thrombocytopenic purpura.

Nephrotic syndrome.

2.VENOUS SINOUS THROMBOSIS

3.FIBRO MUSCULAR DYSPLASIA:

4.VASCULITIS:

[a]Systemic vasculitis:

Polyarteritis Nodosa

Wegners Granulomatosis

Takayasu arteritis

Giant cell arteritis

[b]Primary CNS vasculitis.

[c]Meningitis[Syphilis, tuberculosis,fungal,bacterial,Zoster]

[d]Cardiogenic:

Mitral valve calcification.

Atrial Myxoma

Marantic Endocarditis.

5.DRUGS

Cocaine, amphetamine

6.ECLAMPSIA

HEMORRHAGIC STROKE:

Aneurysmal subarachnoid hemorrhage is the most important treatable condition followed by hypertensive intracranial hemorrhage.

CAUSES OF INTRA CRANIAL HEMORRHAGE:

Head trauma

Hypertensive Hemorrhage[Putamen,Globus pallidus,Thalamus]

Transformation of prior infarction.

Metastatic brain tumour[lung CA ,melanoma]

Coagulopathy

Drugs[cocaine, amphetamine]

Arterio venous malformations

Aneurysm,

Amyloid angiopathy.

Cavernous angioma.

Dural arterio venous fistula

Capillary Telangiectasias.

RISK FACTORS FOR STROKE:

Identification and control of modifiable risk factors is the best strategy to reduce the burden of the stroke as the total number of strokes could be reduced substantially by these means

ATHEROSCLEROSIS RISK FACTORS:

Older age,Family history of thrombotic stroke,Diabetes mellitus,Hypertension,Tobacco smoking,abnormal blood cholesterol particularly low HDL and high LDL[37,43].

HYPERTENSION is the most significant of risk factors.

STROKE: RISK FACTORS

RISK FACTOR	RELATIVE RISK	RELATIVE RISK REDUCTION WITH TREATMENT.
1.Hypertension	2-5	38%
2.Atrial fibrillation	1.8-2.9	68% warfarin 21%Aspirin
3.Diabetes	1.8-6	No proven effect
4.Smoking	1.8	50% at 1 year Baseline risk at 5 years post cessation
5.Hyperlipidemia	1.8-2.6	10-29%
6.Asymptomatic carotid stenosis	2	53%
7.Symptomatic carotid stenosis[70-99%]		65% at 2 years
8.Symptomatic carotid stenosis[50-69%]		29% at 5 years

PATHOPHYSIOLOGY OF ISCHEMIC STROKE:

Acute occlusion of an intra cranial vessel causes reduction in blood flow to the brain region it supplies.The magnitude of flow reduction is a function of collateral blood flow and this depends on individual vascular anatomy and the site of occlusion.

A fall in cerebral blood flow to

Zero-----Death of brain tissue within 4-10 minutes.

Less than 16-18 ml/100g tissue/minute-----Infarction within an hour

If blood flow is returned prior to a significant amount of cell death,the patient may experience only transient symptoms, called transient ischemic attack.Tissue surrounding the core region of infarction is ischemic but reversibly dysfunctional and it is referred to as the ischemic penumbra. The penumbra may be imaged by using perfusion diffusion imaging with MRI. The ischemic penumbra will eventually infarct if no change in flow occurs and hence saving the ischemic penumbra is the goal of thrombolytic therapy.

CLINICAL FEATURES:

Symptoms of stroke appear suddenly and often there is more than one symptom at the same time.

1.Sudden numbness or weakness of the face ,arm or leg especially on one side of the body.

2.sudden confusion ,difficulty in talking or understanding speech

3.Sudden difficulty in seeing in one or both eyes.

4.Sudden difficulty in walking ,dizziness or loss of balance or coordination.

5.Sudden severe headache with no known cause.

Clinical examination should be focused on the peripheral and cervical vascular system[carotid auscultation for bruits, blood pressure],the heart[dysrhythmia,murmurs],extremities [peripheral emboli] and retina[effects of

hypertension and cholesterol emboli]

Disabilities that can result from a stroke include paralysis,cognitive deficits,speech problems,emotional difficulties,fatigue and daily living problems. Many stroke patients requires psychiatric help for post stroke disabilities like depression,anxiety,frustration and anger.Because stroke survivors often have complex rehabilitation needs, progress and recovery are unique for each person.

DIFFERENTIAL DIAGNOSIS:

1.Post ictal Todd's paresis following seizures.

2.Brain tumours may present with acute neurologic symptoms due to hemorrhage,seizures,or hydrocephalous

3.Migraine can mimic cerebral ischemia,even in patients without a significant migraine history. When it develops without head pain [acephalgic migraine],the diagnosis may remain elusive.The diagnosis of migraine becomes more secure as the cortical disturbance begins to cross vascular boundaries or if typical visual symptoms are present,such as scintillating scotomata.

4.Metabolic encephalopathies produce fluctuating mental status without focal neurologic findings.

INVESTIGATIONS:

Once the diagnosis of stroke is made,a brain imaging study is necessary to determine if the cause of stroke is ischemic or hemorrhagic. Computed tomography[CT] imaging of the

brain is the standard imaging modality to detect the presence or absence of intracranial hemorrhage. Early CT scaning of brain will not pick up the infarct but helps to exclude hemorrhage.Infarct may not be seen reliably for 24-48 hours in CT brain.

MRI reliably documents the extent and location of infarction in all areas of brain ,including the posterior fossa and cortical surface. MRI is less sensitive than CT for detecting acute blood.

Other baseline investigations to be done are

Chest X ray

ECG,

Urine analysis,

Complete blood count,

Serum electrolytes ,

Blood urea nitrogen,

Creatinine,

Blood sugar,

Serologic tests for syphilis,

Serum lipid profile

Diffusion weighted imaging is more sensitive for early brain infarction than standard MR sequences ,as is FLAIR[Fluid Attenuated Inversion Recovery] imaging.

Conventional X ray cerebral angiography is the “gold standard” for

identifying and quantifying atherosclerotic stenosis of the cerebral arteries

Paradoxical embolization is identified using bubble contrast echocardiography.It is done by intravenous injection of agitated saline coupled with either transthoracic or trans esophageal echocardiography.It can reveal a cardiac right to left shunt via a patent foramen ovale or atrial septal defect.

TREATMENT:

After the clinical diagnosis of stroke is made, an orderly process of evaluation and treatment should follow.The first goal is to prevent or reverse brain injury . After initial stabilization, an emergency noncontrast CT scan brain should be performed to differentiate ischemic from hemorrhagic stroke.There are no reliable clinical findings that conclusively separate ischemia from hemorrhage, although a more depressed level of conciousness and higher initial blood pressure favour hemorrhage,and a deficit that remits suggests ischemia.

Treatment designed to reverse or lessen the amount of tissue infarction fall within five categories:[36]

1.Medical support.

2.Thrombolysis

3.Anticoagulation

4.Antiplatelet agents

5.Neuroprotection.

1.Medical support:

When cerebral infarction occurs, the immediate goal is to optimize cerebral perfusion in the surrounding ischemic penumbra.Prevent the common complications of bed ridden patients like infections[pneumonia, UTI and skin infections] and deep vein thrombosis with pulmonary embolism.Pneumatic compression stockings prevent deep vein thrombosis. Subcutaneous heparin appears to be safe as well.

Blood pressure should be lowered if there is malignant hypertension, or concomitant myocardial ischemia or if blood pressure is more than 185/110 mmHg and thrombolytic therapy is anticipated.Fever and hyperthermia are detrimental and should be kept under control.

Cerebral edema peaks on the second or third day to cause obtundation or brain herniation. The larger the infarct,the greater the likelihood that clinically significant edema will develop.Even small amount of cerebellar infarction with edema acutely increases intracranial pressure.Treat with water restriction and IV mannitol.

2.Thrombolysis:

If the stroke is ischemic, administration of tissue plasminogen activator may be beneficial in restoring cerebral perfusion. IV r tpa 0.9 mg/kg 10% as bolus, remainder over 60 minutes is given for stroke patients within 3 hours of onset [31]

3.Anti platelet agents:

Aspirin when used within 48 hours of stroke onset reduced both stroke recurrence risk and mortality. [35,42]

4.Anti coagulation:

Heparin given subcutaneously afforded no additional benefits over aspirin and increases bleeding rate in ischemic stroke.[32]

Anti thrombotic prophylaxis in atrial fibrillation:

AGE	RISK FACTORS	RECOMMENDATION
<65 years	1 or more risk factors 0	Warfarin INR 2-3 Aspirin or no treatment
65-75 years	1 or more risk factors 0	Warfarin INR 2-3 Warfarin INR 2-3 or aspirin
>75 years		Warfarin INR 2-3

Risk factors include previous transient ischemic attack or stroke,hypertension,heart failure,diabetes,clinical coronary artery disease,mitral stenosis,prosthetic heart valves or thyrotoxicosis[34,38,39,41]

5.Neuroprotection:

Neuroprotection is the concept of providing a treatment that prolongs the brain's tolerance to ischemia. Hypothermia may be useful.[40]

CAROTID BIFURCATION ATHEROSCLEROSIS:

It is the most common source of artey to artery embolus and specific treatments have proven efficacy in reducing risk.Carotid atherosclerosis produces an estimated 5% of ischemic stroke and the risk of stroke rises, the higher the degree of carotid narrowing.Carotid disease can be classified by whether the stenosis symptomatic or asymptomatic and by the degree of stenosis[33].

Treatment:

Carotid endarterectomy

Balloon angioplasty coupled with stenting.

TREATMENT OF INTRA CRANIAL HEMORRHAGE:

In patients with acute sub arachnoid hemorrhage, blood pressure should be lowered to a normal range with nonvasodilating agents such as Nicardipine,labetolol or esmolol .Patients with cerebellar hemorrhage or with depressed mental status and radiographic evidence of hydrocephalous should undergo urgent neurosurgical evaluation.

Stuporous or comatose patients generally are treated presumptively for elevated intracranial pressure,with tracheal intubation and hyperventilation,mannitol administration and elevation of the head end of the bed while surgical consultation is obtained.

REHABILITATION:

It improves neurologic outcomes and reduces mortality .Proper rehabilitation of the stroke patient includes early physical,occupational and speech therapy.The goal of rehabilitation is to return the patient to home and to maximize recovery by providing a safe,progressive regimen suited to the individual patient.

DEPRESSION FACTS:

Mood disorders are characterized by a disturbance in the regulation of mood, behaviour and affect.Mood disorders are subdivided into

1.Depressive disorders

2.Bipolar disorders

3.Depression in association with medical illness or substance abuse.

INTRODUCTION:

Depression is a serious medical condition that affects thought, feelings and the ability to function in everyday life. It can occur at any age. NIMH [National Institute of Mental Health] sponsored studies showed 10% of American adults have experience some form of depression every year.An interaction between genetic predisposition and life style appears to determine a persons level of risk for depression. Episodes of depression may then be triggered by stress, difficult life events, side effects of medications or other environmental factors.

In the global burden of disease study conducted by WHO , unipolar major depression ranked fourth among all diseases in terms of DALY(Disability Adjusted Life Years)and was projected to rank second by year 2020.

DEPRESSION IN ASSOCIATION WITH MEDICAL ILLNESS:

1.CARDIOVASCULAR SYSTEM:

20 to 30% of cardiac patients(Unstable angina, Myocardial infarction, Cardiac transplant)

2.MALIGNANCY:

The prevalence of depression is 25% in cancer patients commonly with pancreas and oropharaynx cancer.

3.CENTRAL NERVOUS SYSTEM:

Neurological disorders like cerebro vascular disease, Parkinson's disease, Dementia, Multiple sclerosis and traumatic brain injury are associated with depression.

4.DIABETES MELLITUS:

Depression is seen in 8 to 27% of diabetic patients with severity of mood state correlating with the level of hyperglycemia and the presence of diabetic complications.

5.ENDOCRINE:

Hypothyroidism is commonly associated with depression.

6. INFECTIONS:

Life time prevalence of depression in HIV positive individuals is 22 to 45%.Chronic Hepatitis C infection is also associated with depression which may worsen with Interferon alpha treatment.

Depressed patients often show decreased variability in heart rate[an index of reduced parasympathetic nervous system activity] and may predispose individuals to ventricular arrhythmia and increased morbidity .Depression also increases the risk of coronary artery disease due to increased serotonin induced platelet aggregation.

CLINICAL MANIFESTATIONS:

MAJOR DEPRESSION is defined as depressed mood on a daily basis for a minimum duration of 2 weeks. Patient with depression have a profound loss of pleasure in all enjoyable activities, exhibit early morning awakening and often notice a diurnal variation in mood(worse in morning hours).Approximately 15% of population experiences a major depressive episode at some point in life.Depression is often undiagnosed and even more frequently it is treated inadequately.4 to 5% of depressed patients commits suicide.

The term MINOR DEPRESSION is used for individuals who experience atleast 2 depressive symptoms for 2 weeks.

Depression is common in women and increases with age in both sexes

Physicians should also asses the risk of suicide by direct questioning,as patients are often reluctant to verbalize such thoughts without prompting. If significant risk factors exists, the patient must be referred to a mental health specialist for immediate care.

Dysthymic disorder consists of a pattern of chronic [at least 2 years] ongoing,mild depressive symptoms that are less severe and less disabling than those found in major depression.These two conditions can occur together called ‘Double depression”.

Negative life events can precipitate and contribute to depression, but genetic factors influence the sensitivity of individuals to these stressful events.In most cases ,both biologic and psychosocial factors are involved in the precipitation and unfolding of depressive

episodes.The most important stressors appear to involve death of a relative, assault, or severe marital or relationship problems.

Unipolar depressive disorders usually begin in early adulthood and recur episodically over the course of a life time.The best predictor of future risk is the number of past episodes.The duration of an untreated episode varies greatly,ranging from a few months to more than a year.The pattern of recurrence and clinical progression is variable.In a minority of patients, a severe depressive episode may progress to a psychotic state .In elderly patients, depressive symptoms may be associated with cognitive deficits mimicking dementia[pseudo dementia] .A seasonable pattern of depression ,called seasonal affective disorder, may manifest with onset and remission of episodes at predictable times of the year.

SYMPTOMS OF DEPRESSION:

1.Persistent sad, anxious or empty mood.

2.Thoughts of death or suicide or suicide attempts.

3.Feeling of hopelessness, pessimism

4.Feeling of guilt, worthlessness, hopelessness.

5.Loss of interest or pleasure in hobbies and activities that were once enjoyed, including sex,.

6.Decreased energy, fatigue, being slowed down.

7.Difficulty in concentrating, remembering, and making decisions.

8.Restlessness, irritability

9.Insomnia, early morning awakening, or oversleeping.

10.Appetite or weight changes.

If 5 or more symptoms are present everyday for atleast 2 weeks and interfere with daily routine activities, seek an evaluation for depression.

ETIOLOGY:

Although evidence for genetic transmission of unipolar depression is not as strong as in bipolar disorder,monozygotic twins have a higher concordance rate [46%] than dizygotic siblings[20%].A functional polymorphism in the serotonin transporter[5-HTT] gene may interact with stressful life events to markedly increase the risk of depression and suicide. PET studies show decreased metabolic activity in the caudate nuclei and frontal lobes in depressed patients that returns to normal with recovery.

Altered noradrenergic activity , including increased binding to alpha1,2 and beta adrenergic receptors in the cerebral cortex and decreased numbers of noradrenergic neurons in the locus ceruleus is noticed in brains of depressed patients.Involvment of serotonin system is suggested by findings of reduced plasma tryptophan levels, and decreased CSF levels of 5 hydroxy indole acetic acid.

TREATMENT:

Treatment planning requires coordination of short term symptom remission with long term maintenance strategies designed to prevent recurrence.The most effective intervention for achieving remission and preventing relapse is medication, but combined treatment,incorporating psychotherapy to help the patient cope with decreased self esteem

and demoralization ,improves outcome.

Compliance of treatment and outcome improves with

1.Increased intensity and frequency of visits during the first 4-6 weeks of treatment

2.Supplemental educational material

3.Psychiatric consultation as needed.

Between 60-70% of all depressed patients respond to any drug chosen,if it is given in a sufficient dose for 6-8 weeks.A rational approach to selecting which antidepressants to use involves matching the patients preference and medical history with the metabolic and side effect profile of the drug.A previous response or a family history of a positive response to a specific antidepressant would suggest that, that drug be tried first.

CLASSIFICATION:

Anti depressants are of two types:

[a]MAO INHIBITORS

[b]TRICYCLIC AND RELATED ANTIDEPRESSANTS

[a] MAO INHIBITORS:

Non selective:

[1]Hydrazines: Phenelzine

:Isocarboxazid

[2]Non-Hydrazine:Tranylcypromine

Selective:

MAO-A: Clorgiline

Moclobemide

MAO-B: Selegiline[Deprenyl]

[b] TRICYCLIC AND RELATED ANTIDEPRESSANTS:

1.Noradrenaline and serotonin reuptake inhibitors:

Imipramine

Amitryptline

Trimipramine

Doxepine

Clomipramine

Dothiepin

2.Noradrenaline reuptake inhibitors:

Nortriptyline

Desipramine

Protriptyline

Amoxapine.

3.Selective serotonin reuptake inhibitors:

Fluoxetine

Fluvoxamine

Paroxetine

4.Atypical antidepressants:

Trazodone

Bupropion

Mianserin

Tianeptine.

Newer compounds with atypical properties:

Venlafaxine

Mirtazapine

Nefazodone

Citalopram

Sertraline

Tricyclic antidepressants are not euphoriants but only antidepressants. Sedative property varies among different compounds.The more sedative ones are suitable for depressed patients showing anxiety and agitation. The less sedative or stimulant ones are better for withdrawn and retarded patients.

Inhibition of Nor adrenaline and serotonin uptake is associated with antidepressant action.Inhibition of serotonin uptake may be responsible for sedation.

In individuals with suicidal ideation, particular attention should be paid to choosing a drug with low toxicity if taken in overdose. The SSRI's and other antidepressants are distinctly safe in this regard.

Tricyclic antidepressants are contraindicated in patients with Bundle branch blocks and TCA induced tachycardia is an additional concern in patients with CCF.

Selective serotonin receptor uptake inhibitor[SSRI] appear not to induce ECG changes or adverse cardiac events and thus are reasonable first line drugs for patients at risk for TCA related complications. SSRI's may interfere with hepatic metabolism of anticoagulants however,causing increased anticoagulation.

The principal disadvantages of TCAs are antihistamine side effects (sedation)

and anticholinergic side effects (constipation, dry mouth, urinary hesitancy, and blurred vision). Severe cardiac toxicity due to conduction block or arrhythmias can also occur but is uncommon at therapeutic levels. TCAs are probably contraindicated in patients with cardiovascular risk factors. Tricyclic agents are lethal in overdose, with desipramine carrying the greatest risk. Prescribing only a 10-day supply may be judicious. Most patients require a daily dose of 150 to 200 mg of imipramine or amitriptyline or its equivalent to achieve a therapeutic blood level of 150 to 300 ng/mL and a satisfactory remission; some patients show a partial effect at lower doses. Geriatric patients in particular may require a low starting dose and slow escalation.

Second-generation antidepressants include amoxapine, maprotiline, trazodone, and bupropion. Amoxapine is a dibenzoxazepine derivative that blocks norepinephrine and serotonin reuptake and has a metabolite that shows a degree of dopamine blockade. Long-term use of this drug carries a risk of tardive dyskinesia. Maprotiline is a potent noradrenergic reuptake blocker that has little anticholinergic effect but may produce seizures.

Bupropion is a novel antidepressant whose mechanism of action is thought to involve enhancement of noradrenergic function. It has no anticholinergic, sedating, or orthostatic side effects and has a low incidence of sexual side effects. It may, however, be associated with aversive stimulant-like side effects, may lower seizure threshold, and has an exceptionally short half-life, requiring multiple dosing. An extended-release preparation is available.

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SSRIs such as fluoxetine, sertraline, paroxetine, and citalopram cause a lower frequency of anticholinergic, sedating, and cardiovascular side effects but a possibly greater incidence of gastrointestinal complaints, sleep impairment, and sexual dysfunction than do TCAs.

Akathisia, involving an inner sense of restlessness and anxiety, may also be more common, particularly during the first week of treatment. A serious concern, aside from drug interaction, is the risk of "serotonin syndrome," thought to result from hyperstimulation of brainstem $5HT_{1A}$ receptors and characterized by myoclonus, agitation, abdominal cramping, hyperpyrexia, hypertension, and potentially death. Combinations of serotonergic agonists should be monitored closely for this reason. Considerations such as half-life, compliance, toxicity, and drug-drug interactions may guide the choice of a particular SSRI.

Fluoxetine and its principal active metabolite, norfluoxetine, for example, have a combined half-life of almost 7 days, resulting in a delay of 5 weeks before steady-state levels are achieved and a similar delay for complete drug excretion once its use is discontinued. All the SSRIs may impair sexual function, resulting in diminished libido, impotence, or difficulty in achieving orgasm. Sexual dysfunction frequently results in noncompliance and should be asked about specifically in patients using SSRIs. Sexual dysfunction can sometimes be ameliorated by lowering the dose, by instituting drug holidays over the weekend (two or three times a month), or by treatment with amantadine (100 mg tid), bethanechol (25 mg tid), or buspirone (10 mg tid).

Paroxetine appears to be more anticholinergic than either fluoxetine or sertraline, and sertraline carries a lower risk of producing an adverse drug interaction than the

other two. Rare side effects of SSRIs include vasospastic angina and alterations of prothrombin time. Citalopram is the most specific of currently available SSRIs and appears to have no specific inhibitory effects on the P450 system. Escitalopram is the most specific of currently available SSRI's and appear to have no specific inhibitory effects on the p450 system.

Venlafaxine, like imipramine, blocks the reuptake of both norepinephrine and serotonin, but it produces relatively little in the way of traditional tricyclic side effects. Unlike the SSRIs, it has a relatively linear dose-response curve. Patients should be monitored for a possible increase in diastolic blood pressure, and multiple daily dosing is required because of the drug's short half-life. An extended-release form is available and has a somewhat lower incidence of gastrointestinal side effects.

Nefazadone is a selective $5HT_2$ receptor antagonist that also inhibits the presynaptic reuptake of serotonin and norepinephrine. Its side effects are similar to those of the SSRIs, and twice-daily dosing produces a steady state within 4 to 5 days. The drug is related structurally to trazodone, which is currently used more for its sedative than its antidepressant properties. Nefazadone appears to produce a lower incidence of sexual side effects than do the SSRIs.

Mirtazapine is a tetracyclic antidepressant that has a comparatively unique spectrum of activity. It increases noradrenergic and serotonergic neurotransmission through a blockade of central a_2-adrenergic auto- and heteroreceptors and postsynaptic $5HT_2$ and $5HT_3$ receptors. It is also strongly antihistaminic and, as such, may produce sedation at lower doses.

With the exception of citalopram, each of the SSRIs, as well as nefazadone, may inhibit one or more cytochrome P450 enzymes. Depending on the specific isoenzyme involved, the metabolism of a number of concomitantly administered medications can be dramatically affected. Fluoxetine and paroxetine, for example, by inhibiting 2D6, can cause dramatic increases in the blood level of type 1C antiarrhythmics, while sertraline and nefazadone, by acting on 3A4, may alter blood levels of terfenadine, carbamazepine, and astemizole. Because many of these compounds have a narrow therapeutic window and can cause iatrogenic ventricular arrhythmias at toxic levels, the possibility of an adverse drug interaction should be considered.

Other treatment options include the MAOIs and electroconvulsive therapy. The MAOIs are highly effective, particularly in atypical depression, but the risk of hypertensive crisis following intake of tyramine-containing food or sympathomimetic drugs makes them inappropriate as first-line agents. Common side effects include orthostatic hypotension, weight gain, insomnia, and sexual dysfunction. MAOIs should not be used concomitantly with SSRIs, because of the risk of serotonin syndrome, or with TCAs, because of possible hyperadrenergic effects.

Electroconvulsive therapy is at least as effective as medication, but its use is reserved for treatment-resistant cases and delusional depressions.Transcranial magnetic stimulation[TMS] is an investigational treatment of depression that has been shown to have efficacy in several controlled trials.

Regardless of the medication chosen, the treatment response should be evaluated

after approximately 2 months of therapy. Three-quarters of patients show an adequate response by this time, but if remission is inadequate, the patient should be questioned about medication compliance, and an increase in dose should be considered if side effects are not troublesome. If there is no improvement, consultation with or referral to a mental health specialist is advised. Strategies for treatment then include selection of an alternative drug, combinations of antidepressants, and/or adjunctive treatment with other classes of drugs, including lithium, thyroid hormone, and dopamine agonists. Patients whose response to an SSRI disappears over time may benefit from the addition of buspirone (10 mg tid) or pindolol (2.5 mg tid) or small amounts of a tricyclic antidepressant such as desipramine (25 mg bid or tid). Once significant remission is achieved, drug treatment should be continued for at least 6 to 9 months to prevent relapse. In patients who have had two or more episodes of depression, indefinite maintenance treatment should be considered.

It is essential to counsel patients about depression and the medications they are receiving. An educational approach is best, describing what is known about the depressive syndrome and how the medications may help. Advice about stress reduction, side effects, and expected length of treatment and cautions that alcohol may exacerbate depressive symptoms and impede drug response are helpful. Patients should be given time to describe their experience and the impact it has had on them, their family, and their outlook.

DEPRESSION AND STROKE:

The common behavioural and cognitive sequelae of stroke include depression, psychosis, anxiety, personality change, aphasia and dysprosody among others[23]. Depression following stroke is one of the most under recognized complication of a stroke. Appropriate diagnosis and treatment of depression improves the medical status,enhance their quality of life and reduce their pain and disability .The development of depression after a stroke is a serious condition that can have negative effects on thought, emotions and overall daily functioning, particularly in the first year following stroke. Post stroke depression is independent of functional disability, and cerebrovascular risk factors(21).Slowness and psychomotor retardation is common in post stroke depression{18,24}.The Diagnostic and statistical manual-4 distinguishes major depressive disorder[idiopathic depression] and mood disorder due to general medical conditions.

Mimickers:

Post stroke emotionalism[12], reported in about 10%of stroke patients[House et al 1989]is an abnormal lability of mood during which the patient laughs or cries for no easily discernible reactions. Typically the patient does not feel the expected emotions associated with this outward reactions.

Apathetic symptoms may be difficult to distinguish from depression. Depression is characterized by a primary disorder of mood while apathy is largely a disorder of motivation. Most depressed patients report a subjective sense of unpleasant, dysphoric mood but the apathetic patient often appears flat, shallow and emotionally unconcerned.[27]

INCIDENCE AND DURATION:

The prevalence rates for post stroke depression varies enormously [from 5% to 50%] depending on several factors, including the criteria used to diagnose depression, area of involvement , and how long after the stroke the assessment is made[7,26].

Around 70 to 80% of patients with stroke survives. Of those patients who survive the stroke, major depression occurs in about 20% of patients. Major depression is the most severe form of clinical depression that we recognize in neuropsychiatry. Another, about 20% of the patients will develop minor depression. Most depression occur within the acute period after the stroke[1,6,24]

Of the 600,000 American men and women who experience a first or recurrent stroke each year, an estimated 10 to 27% experience major depression[2] . An additional 15 to 40% experience some symptoms of depression within 2 months following a stroke.[3]

The community based Framingham study diagnosed depression in 47% of 6 month stroke survivors. In a population based cohort of Swedish stroke patients whose mean age was 73years,the prevalence of major depression was 25% at hospital discharge,30%at 3 months after stroke, 16% at 1 year[19]

The average duration of major depression in people who have suffered a stroke is just under a year.[18].

AGE

Younger stroke victims are more likely to become depressed than older stroke victims. [6]

SEX:

Epidemologic data from around the world demonstrate that major depression is more

common in women than in men[Weissman and olfson 1995]. The same relationship between depression and female gender is also found in stroke patients.[Anderson et al 1995, Herrmann et al 1998,Kotila et al 1998,Paradiso and Robinson 1998]

HEMISPHERE AND AREA:

The frontal and left hemisphere stroke are strongly associated with depression[8]. A large MRI study from Finland has reported a significant increase in depression following left hemisphere and frontal stroke.[9].MRI has greater accuracy than CT and frontal subcortical circuits are recognized as the neuroanatomical substrate of much of the depression syndrome[10]

There is significant inverse correlation between severity of depression and distance of the lesion from the frontal pole with left hemisphere stroke.[When compared to right hemisphere stroke][22].The frontal lobe and subcortical nuclei are richly innervated by monoaminergic amines serotonin and norepinephrine. Noradrenergic and serotonin cell bodies originate in the brain stem and send axonal projection through the median fore brain bundle to the frontal lobe.Anterior and subcortical lesions could interrupt these ascending fibres to a greater extent than posterior and more distally placed lesions., resulting in a greater likelihood of Post stroke depression.[24].

Serotonin binding was higher in right hemisphere injury than left hemisphere injury.This relative inability of the left anterior hemisphere to upregulate serotonin receptors after stroke produces depression.[24]

VASCULAR DEPRESSION:

Depressive symptoms is not an inevitable reaction to the effects of a stroke. But

depression is a separate illness that can and should be treated, even when a person is undergoing post stroke rehabilitation. Depression is caused by biological factors provoked by brain injury.

At 1997, Alexoplus and Krishnan coined the term “VASCULAR DEPRESSION” to describe depression associated with stroke. Vascular depression includes both Post stroke depression and MRI detected cerebral infarction in which there are no clinical symptoms such as focal neurological deficit. When the infarct obstructs neuronal network related to mood, the patient is predisposed to depression. Neurological factors are more prominent than genetic factors or psychosocial stress in patients with vascular depression.[20]

Folstein et al found a significantly increased rate of depression in stroke patients compared to orthopedic patients despite similar levels of disability, suggesting that mood dysregulation is a more specific complication of stroke rather than a general response to disability.[24].

EFFECT OF DEPRESSION:

Stroke survivors who are also depressed may be less compliant with rehabilitation, more irritable and may experience personality change[3].Patients with Post stroke depression have lower functional status, increased cognitive impairment and higher mortality rates than stroke patients without depression.[13,24].

Parikh et al noted the depressed patients were significantly more impaired in physical and language functioning 2 years after stroke[25].Morris et al also found 3 fold greater mortality in the depressed group independent of age, sex, type of stroke, lesion

location and size.[14].

TREATMENT:

Both nortryptline and citalopram have been demonstrated in controlled studies to be effective in treating post stroke deoression. Treating post stroke depression, not only improve the patients mental state that is their mood but it will also improve their physical recovery and their cognitive and intellectual recovery from the stroke[6].Those treated for depression for 3 months after stroke had longer survival times[15]

Tricyclic antidepressants[TCA] Nortryptline produces greater reduction in depression compared to Selective serotonin receptor uptake inhibitor[SSRI] Fluoxetine[16].However TCA have significant negative effects on cardiovascular diseases while SSRI are safe in depression in cardiovascular disease[17].The greater safety of SSRI in cardiovascular disease is probably because of their inhibitory effects on platelet activation.The SSRI are also very likely to be safer in stroke disease than TCA and this improved safety profile probably makes them the treatment of choice for most people with Post stroke depression.

MATERIAL AND METHODS

This descriptive study is carried out in Stanley medical college hospital during the period of November 2004 to October 2005.The stroke patients in both gender was taken for evaluating the incidence of depression after stroke. The hemisphere of the brain commonly associated with depression is also noted Totally 60 stroke patients were evaluated for depression.

Inclusion criteria:

Stroke within the last 30 days

Exclusion criteria:

1.Aphasic patients

2.Unconcious patient.

3.Heart,Respiratory,kidney,or liver failure

4.Severe disabling musculoskeletal disorder or cancer.

5.Diagnosis of neuro degenerative disorders such as Parkinsons disease,Alzeimer's disease,Multiple system atrophy or Huntington's disease

6.Pre-existing dementia

7.Recurrent unipolar or Bipolar disorder prior to the stroke.

8.Patient taking drugs prone for depression like beta

blockers,Glucocorticoids,anti convulsants,anti parkinsonian medications.

The disability of the stroke is divided into hemiparesis [more than the power 3] and hemiplegia [less than or equal to 3] to evaluate whether the disability is associated with depression.

The quantitative assessment of power can be done by grading the muscle power as suggested by the **medical research council.**

Grade 5-------Normal power

Grade 4--------Movement against resistance

Grade 3--------Movement against gravity.

Grade 2------Gravity eliminated movement.[Lateral movements in bed]

Grade 1---------There is visible or palpable flicker of contraction but no resultant movement of joint

.

CRITERIA USED TO DIAGNOSE DEPRESSION

DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS, IV Edition

1.Depressed mood most of the day, nearly every day, as indicated by either subjective report[eg….feels sad or empty] or observation made by others[eg….appears tearful]

2.Reccurrent thoughts of death[not just fear of dying],recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

3.Feelings of worthlessness or excessive or inappropriate guilt[which may be delusional]nearly everyday[not merely self reproach or guilt about being sick]

4.Markedly diminished interest or pleasure in all, or almost all , activities most of the day, nearly every day[as indicated by either subjective account or observation made by others] .

5.Fatigue or loss of energy nearly every day.

6.Diminished ability to think or concentrate or indecisiveness, nearly every day[either by subjective account or as observed by others]

7.Psychomotor agitation or retardation nearly every day[Observable by others,not merely subjective feelings of restlessness or being slowed down]

8.Insomnia or hypersomnia nearly everyday

9.Significant weight loss when not dieting or weight gain or decrease or increase in appetite nearly everyday.

Five or more of the above symptoms have been present during the same 2 week period and represent a change from previous functioning is needed to diagnose major depression. The symptoms cause clinically significant distress or impairment in social ,occupational, or other important areas of functioning.

The stroke patients are evaluated for depression for 2 weeks using the above Diagnostic and statistical manual IV criteria.The hemisphere side associated with depression is noted.The post stroke depression association with patient's age, sex, hypertension, diabetes ,smoking, alcohol,disability is also studied

STATISTICAL METHOD:

All the data obtained were statistically analysed by using Pearson's Chi square test and the values were considered significant if the p value is <0.05 .

RESULTS

POST STROKE DEPRESSION : INCIDENCE

60 stroke patients were selected for evaluation of depression. Unconcious patients, aphasic patients, patients with respiratory, kidney, and liver failure were excluded from study. Patients with severe disabling musculo skeletal disorder, cancer, neurodegenerative disorders, pre-existing dementia ,recurrent unipolar or bipolar disorder prior to the stroke were also excluded. Stroke patients presenting within 30 days were selected for study.

Total cases taken for study: 60

Patients with post stroke depression: 16 [26.7%]

Patients without post stroke depression: 44 [73.3%]

Out of the 60 cases selected, 16 patients had post stroke depression.26.7% of stroke patients had post stroke depression

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POST STROKE DEPRESSION: ASSOCIATIONS

AGE AND POST STROKE DEPRESSION:[PSD]

	NO PSD	PSD PRESENT	Total
Age less than 40 years	2[66.7%]	1[33.3%]	3
Age 40 to 60 years	31[70.5%]	13[29.5%]	44
Age more than 60 years	11[84.6%]	2[15.4%]	13
Total	44[73.3%]	16[26.7%]	60

	NO PSD	PSD PRESENT	TOTAL
Age less than or equal to 60 years	33[70.2%]	14[29.8%]	47
Age more than 60 years	11[84.6%]	2[15.4%]	13
Total	44[73.3%]	16[26.7%]	60

The younger age patients were commonly associated with depression. Around 30% of stroke patients with age less than or equal to 60 years were associated with post stroke depression while around 15%

of patients with age more than 60 years were associated with depression.It is statistically insignificant. [P=0.57]

SEX AND POST STROKE DEPRESSION:[PSD]

P=0.49

	NO PSD	PSD PRESENT
Male	29 [76.3%]	9[23.7%]
Female	15[68.2%]	7[31.8%]
TOTAL	44	16

HYPERTENSION AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT
NON-HT	22[75.9%]	7[24.1%]
HT	22[71.0%]	9[29%]
TOTAL	44	16

:
P=0.67

DIABETES AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT
NON DM	37[75.5%]	12[24.5%]
DM	7[63.6%]	4[36.4%]
TOTAL	44	16

P=0.42

ISCHEMIC HEART DISEASE AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT
NO IHD	39[70.9%]	16[29.1%]
IHD	5[100%]	0
TOTAL	44	16

P=0.16

SMOKING AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT
NON SMOKER	25[69.4%]	11[30.6%]
SMOKER	19[79.2%]	5[20.8%]
TOTAL	44	16

P=0.4

ALCOHOL AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT
NONALCOHOLIC	27[69.2%]	12[30.8%]
ALCOHOLIC	17[81%]	4[19%]
TOTAL	44	16

P=0.33

HEMISPHERE SIDE AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT	Total
Right hemisphere	29[82.9%]	6[17.1%]	35
Left hemisphere	15[60%]	10[40%]	25
Total	44[73.3%]	16[26.7%]	60

P=0.05.

The **left hemisphere** stroke showed increased incidence of depression compared to right hemisphere stroke,which is statistically significant[p=0.05]. The left hemisphere stroke showed around 40% of depression while right hemisphere stroke showed 17% post stroke depression.

DISABILITY AND POST STROKE DEPRESSION:

	NO PSD	PSD PRESENT
HEMIPARESIS	31[73.8%]	11[26.2%]
HEMIPLEGIA	13[72.2%]	5[27.8%]
TOTAL	44	16

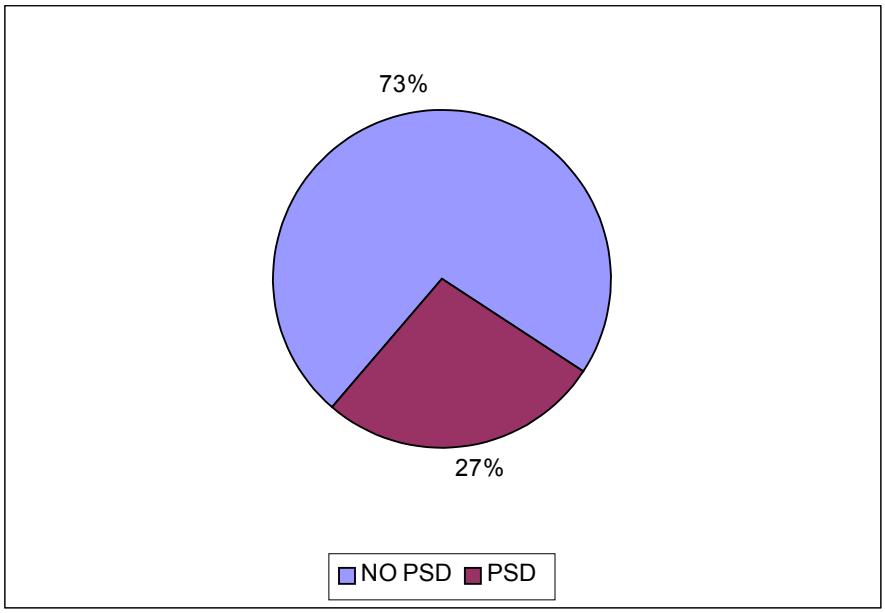
TYPE OF CVA AND POST STROKE DEPRESSION:

	NO PSD	**PSD PRESENT**
INFARCT	40[71.4%]	16[28.6%]
HEMORRHAGE	4[100%]	0
TOTAL	44	16

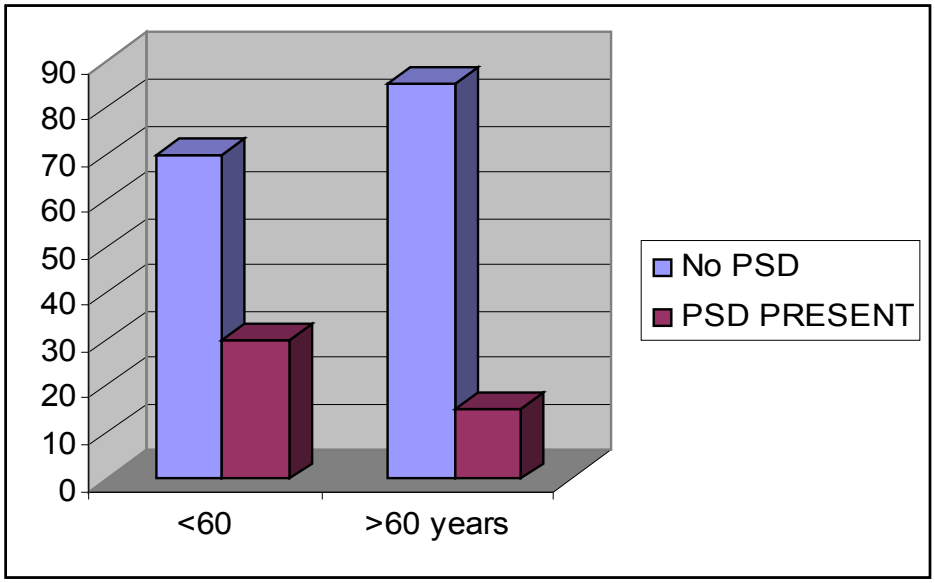
P=0.21

Association of post stroke depression with age, sex, hypertension, diabetes, ischemic heart disease, alcohol, smoking, hemisphere side, disability were studied. Female and male patients had 31.8% and 23.7% of post stroke depression respectively which is statistically insignificant. Hypertension ,Ischemic heart disease, diabetes ,smoking, alcohol association with post stroke depression is statistically insignificant. Left hemisphere stroke patients showed statistically significant depression. [P=.05]

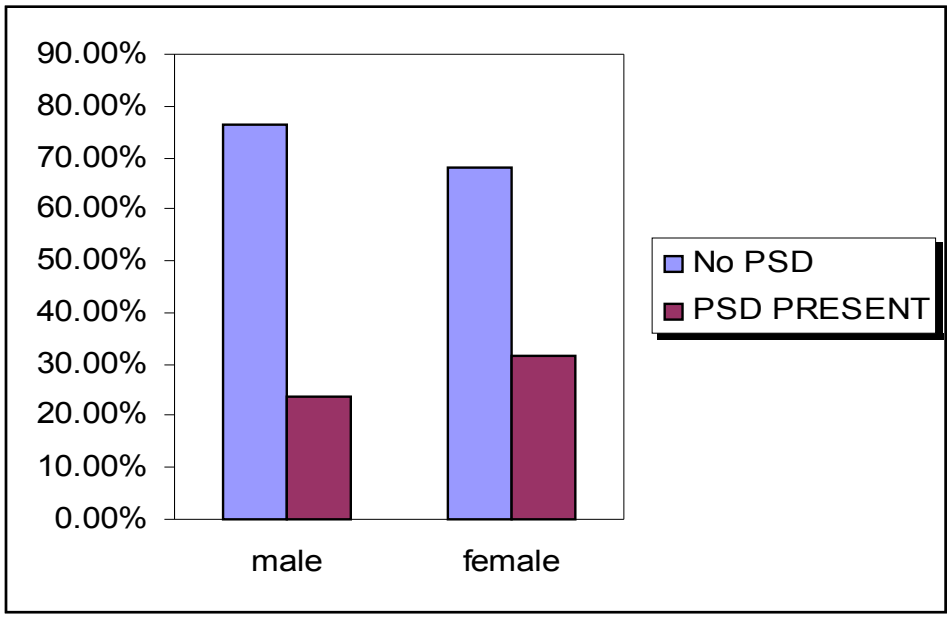
INCIDENCE OF POST STROKE DEPRESSION



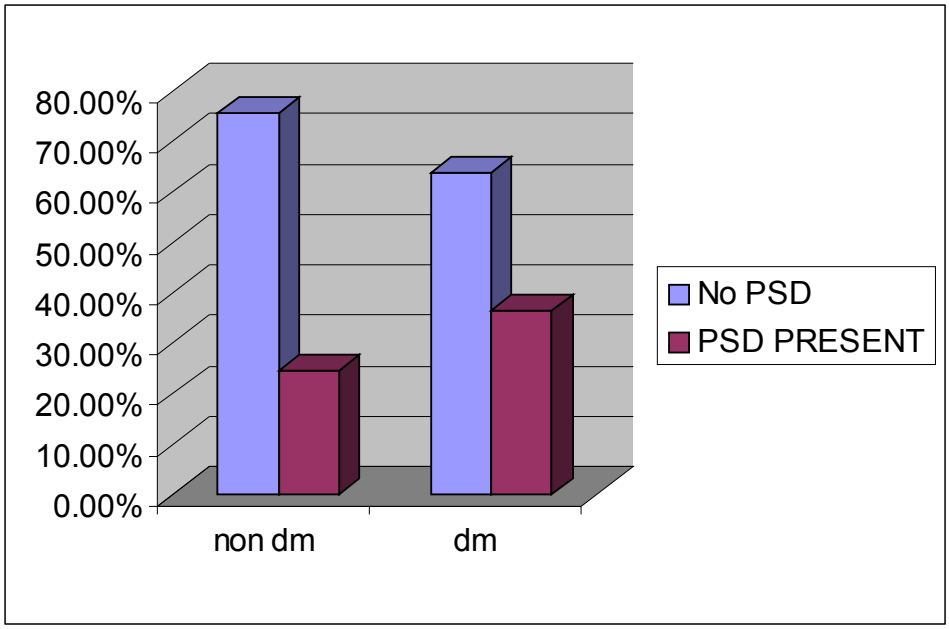
AGE AND POST STROKE DEPRESSION



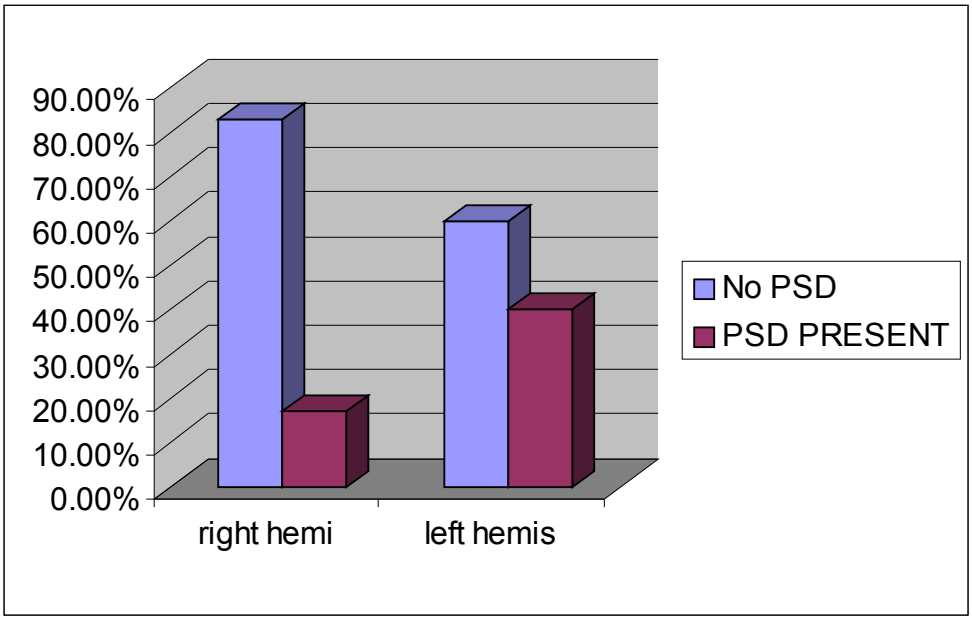
SEX AND POST STROKE DEPRESSION



DIABETES AND POST STROKE DEPRESSION



HEMISPHERE SIDE AND POST STROKE DEPRESSION



PSD – Post Stroke Depression

. **DISCUSSION**

INCIDENCE:

In this study ,60 stroke patients were taken for evaluation of depression.38 cases of male stroke patients and 22 cases of female stroke patients were studied.26.7% of stroke patients were associated with depression.

R Pohjasvaara T, Leppavuori A ,Siira I, Vataja , Kaste M, Erkinjuntti T

in their study found that the prevalence of post stroke depression varies enormously [from 5 % to 50%] depending on several factors, including the criteria used to diagnose depression, area of involvement , and how long after the stroke the assessment is made[7,26].

SEX

Female stroke patients had a increased chance of depression compared to male patients which is comparable to incidence of depression in general population.31.8%of female patients has depression compared to 23.7% of male patients..

[Anderson et al 1995,Herrmann et al 1998, Kotila et al 1998, paradiso and Robinson 1998]

AGE:

The younger age is commonly associated with post stroke depression. Around 30%of patients with age less than or equal to 60 years were commonly associated with depression while around 15% of patients with age more than 60 years were associated with

depression.[6]

[Neau et al 1998,Paradiso and Robinson 1998]

HEMISPHERE SIDE:

The hemisphere side played a major role in determining the depression. Left hemisphere stroke showed increased incidence of depression compared to right hemisphere stroke, which is statistically significant[p=0.05].The left hemisphere stroke showed 40% post stroke depression while right hemisphere stroke showed 17% post stroke depression.[22,24]

Rockville M.D in his study made out the frontal and left hemisphere stroke are strongly associated with depression[8]. A large MRI study from Finland has reported a significant increase in depression following left hemisphere and frontal stroke[9].MRI has greater accuracy than CT and frontal subcortical circuits are recognized as the neuroanatomical substrate of much of the depression syndrome[10]

DISABILITY:

In this study, the disability is not related to depression .The patients with weakness less than or equal to 3 showed 28% of depression while patients with weakness more than 3 showed 26 % of depression. This is statistically insignificant.

At 1997, Alexoplus and Krishnan coined the term "VASCULAR DEPRESSION" to describe depression associated with stroke. Vascular depression includes both Post stroke depression and MRI detected cerebral infarction in which there are no clinical symptoms such as focal neurological deficit. When the infarct obstructs neuronal

network related to mood, the patient is predisposed to depression. Neurological factors are more prominent than genetic factors or psychosocial stress in patients with vascular depression.[20]

Folstein et al found a significantly increased rate of depression in stroke patients compared to orthopedic patients despite similar levels of disability, suggesting that mood dysregulation is a more specific complication of stroke rather than a general response to disability.[24]

DIABETES:

Patients with diabetic has showed increased depression compared to non diabetic. Non diabetic with stroke showed 25% incidence of depression while the diabetic with stroke showed 36%.This is explained by the depression associated with hyperglycemic levels and diabetic complications.[Harrisons]

In this study hypertension, ischemic heart disease, smoking, alcohol did not have significant association with depression in stroke patients.

LIMITATIONS:

The localization of infarct to the particular area in a hemisphere is difficult because of early CT scanning of the brain which will not pick up the infarct area.

This limitation made to limit the study to right or left hemisphere association with depression.

MRI scanning of the brain would help to localize the area of hemisphere concerned with depression. The proximity of infarct to the left frontal lobe determines depression according to the literature.

CONCLUSION

1.27% of stroke patients developed post stroke depression.

2.The left hemisphere stroke is commonly associated with depression.

3.Post stroke depression is common in younger age patients .

4.The severity of disability is not related to depression.

IN SUMMARY, DEPRESSION IS COMMON FOLLOWING THE STROKE AND IDENTIFYING POST STROKE DEPRESSION WILL HELP TO IMPROVE THE PHYSICAL,COGNITIVE AND INTELLECTUAL RECOVERY OF STROKE PATIENTS USING ANTIDEPRESSANTS.

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PROFORMA

Name Age Sex IP number

Complaints

H/O Present illness:

Past History: Hypertension

: Diabetes mellitus

:Ischemic Heart disease

: Heart,Respiratory,Kidney or Liver failure

:Neurodegenative disorders like Parkinsons disease,Alzeimer's disease,Multiple system atrophy,Huntington's disease.

Personal history: Smoking ,

Alcohol

General Examination:

Vital signs: Pulse rate

Blood pressure

Systemic examination:

Central Nervous system:

Higher function: Level of conciousness

Orientation

Intelligence

Memory

Sleep

Speech and language:Dysarthria

Aphasia:Motor,sensory and global aphasia

Cranial nerves:

Motor system: Bulk

Tone

Power

Reflexes

Co-ordination of movements

Gait and involuntary movements

Sensory system: Light touch,position sense,Recognition of size,shape,weight and form,vibration,pain,temperature.

Clinical diagnosis:

CT Diagnosis and other baseline investigations,ECG,CXR

Diagnosis if depression using DSM IV criteria[Diiagnostic and statistical manual]